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The relationship of cognitive and affective factors with daily somatic complaints: An investigation into response expectancy theory and the symptom perception hypothesis

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Abstract

Theories explaining how psychological factors influence somatic complaints have existed for decades; however, few attempts have been made to integrate their predictions. Two such theories are the symptom perception hypothesis and the response expectancy theory. The symptom perception hypothesis suggests that negative affect influences daily somatic complaints, whereas the response expectancy theory surmises relationships with response expectancies. Neurological evidence suggest psychological factors such as these should be integrated to better understand daily somatic complaints. In line with this suggestion, this study aims to examine the combined effects of three psychological factors: namely state negative affect, response expectancies, and recollection of prior symptoms, in predicting daily somatic complaints. Using a sample of 95 college students alongside multilevel modelling, this study examined an integrated model of daily somatic complaints. Daily diary paradigms were used to measure state negative affect and daily somatic complaints. Response expectancies and recalled symptoms were measured as generalized constructs. Additionally, the interaction of state negative affect and response expectancies was examined. Results suggest an additive effect, as state negative affect, response expectancies, and recalled symptoms all predicted daily somatic complaints. The cross level interaction of expectancies and state negative affect was not significant. The results provide support for the coexistence of both the symptom perception hypothesis and response expectancy theory, as well as support for the integration of psychological factors.

Keywords: Somatic complaints, expectancies, affect, recall, symptom

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The relationship of cognitive and affective factors with daily somatic complaints: An investigation into response expectancy theory and the symptom perception hypothesis

The Center for Disease Control (2012) estimated that in 2009, Americans visited a medical setting about 1.3 billion times, and averaged about $8000 per capita expenditure. This totaled around $2.5 trillion spent on medical visits. Somatic complaints, such as headaches, are one of the primary reasons for medical care visits (US Dept. of Health, 2009a, 2009b) and can be used by primary care providers in diagnostic procedures (e.g., differential diagnosis, pattern recognition, etc.) to initially guide diagnostic and medical decisions. Research has demonstrated that this reliance on symptom reporting can lead to large health care expenditures (Kroenke and Mangelsdorff, 1989). Likewise, somatic complaints appear to be associated lower quality of life (Smith, Monsen, and Ray, 1986; Stanley, Peters, and Salamon, 2002) and psychological disorders (Kroenke, 2003; Kroenke, Spitzer, Williams, Linzer, Hahn, deGruy, & Brody, 1994). Although the consequences of reporting normal somatic sensations as symptoms appear to be large, the consequences of misattributing actual symptoms may be even greater. As discussed by Suls and Howren (2012), such misattributions could ultimately end in life or death scenarios. When the symptoms have an organic basis, they can be invaluable sources of information for guiding treatment and diagnosis. Therefore, accurate symptom reporting can have major implications in treatment as well as in costs and daily functioning.

Somatic complaints originate from subjective information generated from within the body (Craig, 2009; Kirsch, 1985), and it has been suggested that psychological factors influence daily somatic complaints (DSC) due to this subjective nature (Kirsch, 1985; Pennebaker, 1982; Rief & Broadbent, 2007; Suls & Howren, 2012). It should be noted though, that in the case of strong situational influences (e.g., obvious physical trauma), such complaints are unlikely to be

unambiguous (Howren & Suls, 2011). This would attenuate any relationships between psychological factors and DSC. Thus, the focus of research relating psychological factors to DSC has focused on mild to moderate DSC that do not necessarily have an identifiable cause. Research has generally supported these linkages (Brown, & Moskowitz, 1997; Cohen, Doyle, Skoner, Fireman, Gwaltney, & Newsom, 1995; Feldlman, Koerner & Kenyon, 2007; Larsen, 1992; Ode, Hilmert, Zielke, Robinson, 2010; Suls & Howren, 2012; van Wijk, Huisman, & Kolk, 1999; Watson, 1988; Williams, & Wiebe, 2000).

Within this literature, gaps in our knowledge exist. First, existing theories typically emphasize the impact of a conscious attending to somatic sensations in the generation of DSC (Pennebaker, 1982; Watson & Pennebaker, 1989); however the body continuously produces somatic information and it is unclear how the body selects which information to bring into conscious awareness (Rief & Broadbent, 2007). Additionally, this corpus of theoretical and research literature has tended to not coalesce. For instance, two theories, symptom perception hypothesis (Watson & Pennebaker, 1989) and response expectancy theory (Kirsch, 1985), arose in different contexts, and their combined predictive capabilities have yet to be adequately investigated in daily life. However, neurological literature suggests a mechanism by which somatic information can be brought into conscious awareness, as well as a means to integrate psychological theories (Craig, Chen, Bandy, & Reiman, 2002; Craig 2003, 2009).

Interoception and Somatic Complaints

Interoception has been defined as a sense of the physiological state within the body (Craig, Chen, Bandy, & Reiman, 2002; Craig 2003, 2009). It is proposed to encompass senses such as nociception, visceral sensation, and chemoreception (Craig et. al, 2002). This

interoceptive process can be conceived as a bodily mechanism that monitors the continuously produced somatic information, and selects which information is transferred to higher cognitive areas. An understanding of what factors influence this transfer of information is key to understanding DSC.

Based on animal and human research, Craig and colleagues (2002, 2003, 2009) proposed that the insular cortex is the primary brain area involved in interoception and self-awareness. Per his model, a primary interoceptive representation of the body is first projected to the posterior insula. This information then proceeds from the posterior toward the anterior insula cortex. While this information travels through the mid-insula, it is re-represented and input is incorporated from other brain areas. For instance, input from the hypothalamus and amygdala add affective information. Additionally, input related to cognition (e.g., expectancies, short-term memory, etc.) is incorporated from the anterior cingulate cortex, ventromedial prefrontal cortex, and the dorsolateral prefrontal cortex. This affective and cognitive information is thought to increase the salience of somatic information, thus increasing the possibility the information is brought into conscious awareness for perceptual processing. Through this process, psychological factors could bring somatic information into conscious awareness.

It is possible that cognitive factors (e.g., response expectancies) increase the salience of somatic information, thus bringing it into conscious awareness; this proposed dual role of psychogenic and somatogenic factors suggests that research integrating markers of cognitive and affective factors associated with the body's stress response (e.g., anxiety) holds promise of elucidating their combined influence on DSC. As such, this study aimed to incorporate two theories from different lines of research, namely the Symptom-Perception Hypothesis (Watson &

Pennebaker, 1989) and response expectancy theory (Kirsch, 1985). Furthermore, this study will investigate the influence of recollection of symptoms experienced previously.

Symptom-Perception Hypothesis.

Negative affect is characterized by subjective distress and negative emotions (Watson, 1988; Watson & Tellegen, 1985). It has been separated into both trait (similar to the personality construct of Neuroticism; Watson & Tellegen, 1989) and state (SNA) processes, and has been found to fluctuate during daily life (Watson, 1988; Watson & Tellegen, 1985). The literature suggests that both trait negative affect and SNA are positively related to somatic complaints (Charles & Almeida, 2006; Jorgensen & Richards, 1989; Watson & Pennebaker, 1989). The symptom perception hypothesis attempts to provide a theoretical basis for the relationship.

The symptom perception hypothesis (Watson & Pennebaker, 1989) originally proposed that negative affect reflects a tendency to attend to, process and inflate the negative valence of internal sensations. One area this theory did not address was how somatic information is selectively brought into conscious awareness via this attentional mechanism. Based on the combined impact of somatogenic and psychogenic factors discussed earlier, it seems reasonable to expect that affective information (e.g., negative affect) is joined with interoceptive processes, thus increasing the affective salience of somatic information, thereby increasing the likelihood of bringing greater somatic information into conscious awareness for processing. This increased likelihood would, in turn, foster greater perception of DSC.

The literature typically suggests that SNA, a correlate autonomic activity, is a consistent, affective predictor of DSC. More specifically, research has demonstrated that SNA is a stronger predictor of DSC compared against trait negative affect (Brown & Moskowitz, 1997; Charles

and Almeida, 2006; Ode, Hilmert, Zielke & Robinson, 2010) and stress (Ode et. al, 2010). Furthermore, SNA has been suggested to mediate gender differences in somatic complaints (van Wijk et. al, 1999), and to partially mediate the relationship with trait negative affect (Williams, & Wiebe, 2000). As such, evidence exists suggesting that SNA is an influential factor in DSC.

Despite this knowledge, gaps exist in current knowledge. One limitation of this research is that SNA has predominantly been investigated in relation to personality and stress, with less of a focus on cognitive factors. As mentioned previously, literature suggest a better understanding of cognitive and affective relationships to DSC would be beneficial (Craig, 2009; Howren & Suls, 2011). This study aims to fill this gap by examining the interrelations of cognition, affect, and DSC.

Response Expectancy Theory

Response expectancies represent subjective probability estimate of the likelihood of occurrence one's non-volitional (automatic) responses to internal and external cues (Kirsch, 1985, 1997, 1999). In describing response expectancies, Kirsch (1997) states that the extent to which they influence interoceptive stimuli is a function of the ambiguity of the stimuli. He suggests the more ambiguous the stimuli, the greater the influence of response expectancies. As previously discussed, somatic sensations, such as DSC, have been proposed to be highly subjective (Craig, 2009; Kirsch, 1985), and as such, would appear to be particularly susceptible to generalized response expectancies (Mischel, 1999).

Based on response expectancy theory (Kirsch, 1985, 1997, 1999), expectancies lead to a self-confirming effect by influencing the perception of internal sensations (Kirsch, 1999). Research suggests that response expectancies could increase the salience of somatic information,

thus bringing this information into conscious awareness (Kong, Gollub, Rosman, Webb, Vangel, Kirsch, & Kaptchuk, 2006; Lorenze, Hauck, Paur, Nakamura, Zimmermann, Bromm, & Engel, 2005; Schmidt, Wolfs-Takens, Oosterlaan, va den Hout, 1994). This process would then cause a greater amount of somatic information to be processed (Kirsch, 1999; Pennebaker, 1982), and lead to the experience of greater sensations and symptoms (Pennebaker 1982). Through this process it is presumed that higher response expectancies leads to higher levels of DSC.

Some support for this presumption can be found in the literature, but this research has been limited to laboratory and treatment settings. For instance, research implicates higher response expectancies in the reporting of more somatic complaints for populations undergoing treatments for smoking (Tate, Stanton, Green, Schmitz, Le, & Marshall, 1994), alcohol abuse (Hawker & Orford, 1998), colposcopy (Kola, Walsch, Hughes, & Howard, 2011) and breast cancer surgery (Montgomery & Bovbjerg, 2004). Further evidence can be found from lab research using a cold presser apparatus (Sullivan, Rodgers, & Kirsch 2001), an infrasound exposure manipulation (Crichton, Dodd, Schmid, Gamble, & Petrie, 2013), and exposure to an environmental "toxin" manipulation (Lorber, Mazzoni, & Kirsch, 2007). This research demonstrates that response expectancies tend to be positively related to somatic complaints; however, it is unclear if this relationship extends beyond the lab and treatment context into normal, daily life.

Despite this literature base, limitations exist in our knowledge. One limitation is that such associations may not show external validity with assessments of mood and expectations associated with everyday life. An understanding on how expectations in everyday life could contribute to generalizing these lab findings to a variety of ecological settings (e.g.,

expectancies' influence on symptom complaints in the primary care, educational or work context).

Furthermore, it seems plausible that recall of prior symptoms (recalled symptoms) could act as a third variable that is driving the relationship between response expectancies and DSC in normal populations (Rief & Broadbent, 2007). In this regard, one would expect that recalled symptoms alter expectations and experiences of that symptom in the future. To illustrate this, let's examine headaches. Recollections of experienced headaches could cause an individual to expect to experience future headaches (i.e., increase headache response expectancies). These recalled symptoms, then, would be present in working memory in the dorsolateral prefrontal cortex and thus could influence the salience of somatic information (Craig, 2009). Thus, recalled symptoms could be related to both response expectancies and DSC. To date, no research has investigated the influence of recalled symptoms. As such, a second aim of this study is to conduct a preliminary investigation into the influence of recalled symptoms, with the expectation that recalled symptoms would be positively related to both symptom response expectancies and DSC.

The Present Study.

The principle aim of this study was to integrate response expectancy theory and symptom perception hypothesis in the prediction of DSC. More specifically, this study was designed to examine the individual and combined influences of response expectancies and SNA on DSC. Furthermore, this study investigated the effects of recalled symptoms on DSC, as well as its influence on the relationships between response expectancies, SNA, and DSC.

As mentioned previously, SNA has been found to fluctuate in daily life, and the literature suggests that SNA is a stronger predictor than trait negative affect As such, a daily diary paradigm was used to measure SNA as well as DSC. Daily diaries have been found to maximize ecological validity, decrease recall bias and capture between and within person variance (Gunthert & Wenze, 2012); this procedure has been used in a similar fashion in previous research (Charles & Almeida, 2006; Hilmert, Ode, Zielke, & Robinson, 2010; Kolk, Hanewald, Schagen, van Wijk, 2003; van Wijk, Huisman, & Kolk, 1999). Furthermore, daily measurement would provide more information in terms of both mean levels and daily variation.

Related to response expectancies, research has shown that expectancies tend to have high temporal stability (r >.67; Catanzaro & Mearns, 1990). Furthermore, as mentioned previously, situational influences (e.g., physical trauma) are thought to remove the subjectivity of somatic sensations, and could provide individuals with definitive, causal explanations. Related to mild to moderate somatic complaints, due to their subjective nature, it would be expected that generalized expectancies would demonstrate greater relationships (Kirsch, 1997; Mischel, 1999). Consequently,response expectancies were measured as a generalized expectancy construct..

Integrating response expectancy theory and symptom perception hypothesis.

The symptom perception hypothesis (Watson & Pennebaker, 1989) and the supporting literature (Brown & Moskowitz, 1997; Charles and Almeida, 2006; Ode, Hilmert, Zielke & Robinson, 2010) suggest SNA is related to DSC. Additionally, it is presumed that affective information (e.g., SNA) from the hypothalamus and amygdala would increase the salience of somatic information (Craig 2003, 2009). Thus, it was expected that SNA would be related to

DSC. In other words, it was expected that, on days in which participants reported higher levels of SNA, they would also report higher levels of DSC.

Response expectancy theory (Kirsch, 1985, 1997, 1999) and the supporting literature (Crichton et. al., 2013; Hawker & Orford, 1998; Kola et. al., 2011; Lorber et. al., 2007; Montgomery & Bovbjerg, 2004; Sullivan et. al., 2001; Tate et. al., 1994) suggest a positive relationship between response expectancies and DSC. Thus it was expected response expectancies would be related to DSC. That is, it was expected that individuals who reported higher levels of response expectancies would report higher levels of DSC. Similarly to SNA, it is expected that response expectancies would increase the salience of somatic information; however, expectancy related information may be generated from a variety of brain regions (Kong et. al., 2006), including anterior cingulate cortex and prefrontal cortex (Craig 2003, 2009; Kong et. al., 2006). Since information is being integrated from different areas of the brain, it was expected that SNA and response expectancies would demonstrate an additive effect in predicting DSC. Mathematically, this model would look like *Equation 1,* for the i^{th} individual on day j.

$$DSC_{ij} = \gamma_{00} + \gamma_{i0}(response\ expectancies) + \gamma_{ij}(SNA) + \mu_{0j} + \varepsilon_{ij} \quad (1)$$

Recently, Charles and Almeida (2006) demonstrated that trait negative affect, a correlate of symptom reporting, moderated the association between SNA and DSC; with SNA and DSC showing the strongest correlation among high trait negative affectivity persons. Recall that the generalized response expectancies also is thought to covary with DSC. Like the individual difference of trait negative affectivity, it is conceivable that the association of SNA and DSC may be moderated by response expectancies. To date, this question has not been examined empirically. Consequently, exploratory analyses were conducted to examine the moderating

effects response expectancies on the relationship between SNA and DSC. Like trait negative affectivity, it is possible SNA and DSC are associated only in the context of high response expectancies individuals due to their greater likelihood to construe internal sensations, both affective physical, in terms of a negative valence. Mathematically, this model would look like *Equation 2*, for the i[th] individual on day j.

$$DSC_{ij} = \gamma_{00} + \gamma_{j0}(response\ expectancies) + \gamma_{ij}(SNA) + \gamma_{ij}(response\ expectancies\ xSNA) + \mu_{0j} + \varepsilon_{ij} \quad (2)$$

The influences of recalled symptoms.

Several exploratory analyses were conducts to investigate the influences of recalled symptoms on DSC and to facilitate understanding the influences of SNA and response expectancies on DSC. If recalled symptoms shape expectations, then it should correlate positively with response expectancies. Furthermore, if recalled symptoms increase the salience of somatic information, it should be related to DSC. As mentioned previously, recalled symptoms, SNA and response expectancies are all thought to increase the salience of somatic information; however, information related to recalled symptoms would be presumed to be incorporated from brain areas related to working memory, namely the dorsolateral prefrontal cortex (Craig, 2009). Although the dorsolateral prefrontal cortex is also a region suspected to incorporate response expectancies, response expectances may also include information from other brain areas as mentioned above (Kong et. al., 2006). Furthermore, these regions are different from those incorporating affective information. Thus, it was expected that response expectancies, recalled symptoms and SNA would demonstrate an additive effect in predicting DSC. Mathematically, this model would look like *Equation 3*, for the i[th] individual on day j.

$$DSC_{ij} = \gamma_{00} + \gamma_{i0}(response\ expectancies) + \gamma_{i0}(recalled\ symptoms) + \gamma_{ij}(SNA) + \mu_{0j} + \varepsilon_{ij} \quad (3)$$

Methods

Participants

The total sample size prior to exclusion was 115. The sample consisted of college students to ensure it was composed of healthy adults. This sample was used for three reason. First, it has been suggested that individuals experience more somatic symptoms with increasing age (e.g., pain), but are more accepting of these somatic experiences and therefore less likely to report them (Hilderink, Collard, Rosmalen, & Voshaar, 2015; Sofaer, 2005). By focusing on adults younger than 65, such reporting biases were presumed to be diminished, thus eliminating age as a confounding variable. Second, a sample of individuals who did not report chronic illness was used. Chronic illness has been demonstrated to influence variables measured in this study (Kokkonen, & Kokkonen, 1995; McAndrew, Mora, Quigley, Leventhal, & Leventhal, 2014), and thus it was suspected chronic illness would confound results. Third, this was an initial investigation into the integration of psychological factors. As such it was determined that if the hypothesis were supported, research on a more representative sample could be conducted. As such, inclusion criteria included:

- Being 18-65 years of age
- The absence of a chronic illness.

It should be noted that we did not inquire about any chronic illnesses participants could possess, and thus could not test any differences between illnesses.

Exclusion occurred if participants did not complete any outcome measures (DSC). This resulted in 20 participants being excluded.[1] The final sample consisted of 95 participants, with a total of 486 data points. Analyses showed that included and excluded participants did not differ in recalled symptoms, t (113) =1.113, p=.268. The average age of the participants was 19.7 years (SD = 2.7, age range: 18-45 years), and was majority female (51.6%) and white/Caucasian (65.3% white/Caucasian; 9.5% African American; 13.7% Asian; 6.3% Hispanic/Latino; 5.3% others).

Materials and Procedures

The following procedures were approved by the Institutional Review Board. Participants took part in the study in exchange for course credit. This study was composed of three parts: a pre-lab portion, an in-lab portion and a seven-day diary portion. The *Pre-Lab Session* assessed recalled symptoms, and occurred online seven days prior to the *In-Lab Session*. The *In-lab Session* was comprised of questionnaires measuring demographics and response expectancies. Online informed consent was obtained before the *Pre-Lab Session,* and written informed consent prior to the *In-Lab Session.* Additionally, at the end of the *In-lab Session* participants were trained on the completion of daily diaries. This training consisted of verbal instructions alongside live demonstration, and was supplemented with written instructions.

Following the in-lab session, participants completed internet-based diaries measuring SNA and DSC for seven consecutive days. These diaries were completed between the hours of 6 pm and 12 am as a focus group revealed this time preferable. Furthermore, other research has determined end of day reporting to be sufficient for initial investigations into within-person

[1] It should be noted that additional analyses were run excluding participants that completed fewer than four of seven possible diaries. These additional analyses resulted in similar results, and the author chose to report results for the entire sample.

processes and measuring negative events (Tennen, Affleck, & Armeli, 2005). Reminders were sent between 12pm and 4pm daily to encourage completion of diary entries and, additionally, if participants had not completed diary reports for more than two days.

Assessment took place using the following self-report questionnaires. All questionnaires were counterbalanced with results failing to demonstrate significant differences between versions (all p's>.05). The following information was collected:

Demographics. Participants were asked their date of birth, gender, and race/ethnicity. Age was calculated by subtracting their date of birth from the day they began the study, and was used as a continuous variable. Gender was dummy coded (female as 0, male as 1). Race/ethnicity was entered in two separate ways. First as a variable encompassing all options to be used in ANOVA analyses to test racial difference between groups. Second, race/ethnicity was dummy coded for endorsement of a specific race (e.g., 0 = race not endorsed, 1 – race endorsed) to be used to analyze racial influences in multilevel analyses. This manner was chosen because sample size amongst races were unequal, and therefore For instance, if an individual responded affirmatively to being Hispanic, they were coded as a 1. This resulted in the creation of six variables representing white, black, Asian, Hispanic, and mixed raced/other race.

Cohen-Hoberman Inventory of Physical Symptoms (CHIPS). The Cohen-Hoberman Inventory of Physical Symptoms (CHIPS) was developed as a measure of self-report somatic complaints (Cohen & Hoberman, 1983). Participants rate how much they are bothered by 33 somatic symptoms (e.g., back pain) on a 5-point scale ranging from "not at all" to "extremely". In college populations, the CHIPS has been found to be correlated with the use of health care

services (r=0.22 and r=0.29) and to demonstrate internal reliability with a Cronbach's alpha of 0.88 (Cohen & Hoberman, 1983). Refer to *Appendix A* for an example of the CHIPS.

For this study, the directions were altered to accommodate the different needs of the sessions. During the *Pre-lab Session,* directions asked participants to report the amount they have been bothered by symptoms over the previous six months (recalled symptoms; Cronbach's Alpha = .90). To measure response expectancies, during the *In-lab Session*, participants were asked to predict how often they expect to be bothered by each of the symptoms over the following seven-day period (Cronbach's Alpha = .90). In addition a foil expectancy item (itchy scalp/head) was included, as a check on specificity. More specifically, it was thought that relationships between response expectancies and DSC would be unique to the items on the CHIPS, and that items reflecting symptoms conceptually unrelated to the DSC measured (e.g., itchy scalp) would not be related. Consequently, this item was not expected to predict the symptoms measured by the CHIPS, as it was conceptually unrelated to the other items in the scale. A similar procedure was used in previous research (Montgomery and Bovbjerg, 2004); however the item was changed, as it was suspected that hearing loss would not be endorsed and produce low variability. For DSC, participants were asked to report the somatic symptoms they were bothered by during the last 24 hours. The DSC items were averaged across the seven days and demonstrated good internal consistency (Cronbach's Alpha = .91). Ratings for each item remained the same throughout assessment periods, using the 5 point scale mentioned above.

Positive and Negative Affect Schedule (PANAS). The Positive and Negative Affect Schedule (PANAS) was constructed to assess positive and negative affect (Watson & Clark, 1999). These measures demonstrates good construct validity, convergent validity with rating from others, is related to other mood measures, and is sensitive to within-person fluctuation in

mood (Watson & Clark, 1999; an extensive review of the psychometric properties of the PANAS are available online). For this study, participants completed the negative affect scale daily, as a measure of SNA. Similarly to DSC, the items were averaged across the seven days to determine internal consistency (Cronbach's Alpha = .80). Directions were altered as indicated for DSC above. Refer to *Appendix B* for an example of the CHIPS.

Data Analyses

To begin, the distributions, descriptive statistics and bivariate correlations were computed to examine relationships at the individual level to determine if they were in hypothesized directions. Due to the nested nature of the data (days within individuals) ordinary least squares regression analyses can increase Type I error rate (Hox, 2010), and cause inaccuracy in parameter estimation (Nezlek, 2012). Consequently, multi-level modeling computed by means of MIXED Proc analysis in SPSS was used for data analytic purposes.

For this study, a two-level model was created. At the first level (within-person variables), days, SNA and DSC were modeled. Within person correlations were predicted; thus it was expected that covariance matrices that allowed for such correlations (e.g., autoregressive) would best fit the data. The second level consisted of between-person variables, namely participant (ID number), response expectancies and recalled symptoms. Participants were modeled as a random effect for within-person variance.

Variables were centered per recommendations of Enders and Tofighi (2007). Level one variables (DSC and SNA) were group mean centered. Group mean centering is done by taking the individuals own mean across days and subtracting it from their scores, and creates a measure reflecting how different that particular day's measurement is from that participant's mean.

Enders and Tofighi (2007) suggest centering within group results in uncorrelated level 1 and level 2 variables. Level 2 variables (response expectancies and recalled symptoms) were grand mean centered as literature suggests (Aiken and West, 1991; Enders and Tofighi, 2007). Specific model specifications can be found in *Table 1*.

Results

To investigate descriptive statistics, demographic differences, and bivariate correlations, daily measures were collapsed within participant, creating a within person average for SNA and DSC. *Table 2* displays descriptive statistics and bivariate correlations, and indicates that significant relationships existed between all variables including response expectancies and recalled symptoms. Analysis of demographic effects revealed no significant differences due to gender, all *t's* (93) < 1.28, all *p*'s >.05, or race/ethnicity, all *F*'s (4, 94) < 1.22, all *p*'s>.05. Furthermore, age was not significantly correlated with any variables, -.10< *r* < .01, all *p*'s > .33.

The within-person correlations of SNA and DSC were analyzed to determine if utilizing MLM was justified. Reference to *Table 3* indicates that significant within-person correlations were present in DSC. This suggests that using a covariance structure that models covariances as zero (e.g., diagonal) would not result in a good fit.

Multilevel Prediction of Daily Somatic Complaints (DSC)

A null model (without any predictors) was run, and it was determined there was significant between-person variance, Wald Z = 13.95, *p* <.001, suggesting the use of MLM was justified. The Intraclass Correlation (ICC) was .284, meaning that 28.4% of the variance in DSC was found between participants. Next, various combinations of covariance structures were tested to determine the best fit. The use of certain covariance structures (e.g., unstructured covariance

matrix) failed to converge on a model. Of the covariance structures that resulted in convergence, the lowest model fit criteria was obtained when modeling days with an autoregressive variance and participants with an identity covariance structures (refer to *Table 4 Model 1*). As such, these covariance structures were used for the remainder of this study. Furthermore, perusal of *Model 1* suggests that, on successive days, DSC tended to correlate highly, $\rho = .55$, $p < .001$. The residual and intercept were significant; also, the intercept is greater than the residual suggesting that most of the variability in this model was between participants. After modeling days with an autoregressive covariance structure, the ICC was .604, suggesting 60.4% of the variance was between-participants.

Integrating response expectancy theory and symptom perception hypothesis

The independent main effects of SNA and response expectancies on DSC were then analyzed. This revealed significant fixed effects for the response expectancies, $B = 0.50$, SE = 0.06, t (92.59) = 8.13, $p < .001$, 95% CI [0.38, 0.63], and SNA, $B = 0.47$, SE = 0.10, t (366.00) = 4.92, $p < .001$, 95% CI [0.28, 0.66] in predicting DSC. Random effects for each variable either did not reach significance, $p > .05$. Next, the ability of the foil item to account for changes in DSC was tested, resulting in non-significant effect, t (89.58) = 0.82, $p = .413$.

Next, a model was created in which response expectancies and SNA were entered simultaneously as predictors of DSC. As indicated in *Table 4* (refer to Model 2), the model fit statistics decreased as compared to the no predictors model (Model 1). Additionally, in this model, SNA and response expectancies both remained significant predictors of DSC. In the second model, the residual and intercept remained significant; however, the residual was greater than the intercept suggesting that most of the variability was with-participants at this point. After

inserting SNA and response expectancies, the ICC was .455, suggesting 45.5% of the variance was between-participants.

Following this, exploratory analyses relating to the hypothesis that response expectancies would moderate the relationship between SNA and DSC was conducted. This moderation was analyzed by inserting a cross-level interaction term (response expectancies x SNA) into the model using SNA and response expectancies to predict DSC. Within the model, the response expectancies x SNA term failed to reach significance, $B = 0.005$, SE = 0.005, t (380.17) = 0.88, $p = .381$, 95% CI [-0.006, 0.016], and was subsequently dropped from the final model.

The influence of prior symptom experience

Reference to *Table* 2 indicates a significant relationship was found between recalled symptoms and response expectancies. Additionally, when modelled to predict DSC, recalled symptoms demonstrated a significant, fixed effect, $B = 0.53$, SE = 0.07, t (90.20) = 7.41, $p < .001$, 95% CI [0.39, 0.67] and random effects, $B = 50.87$, SE = 2223, Wald Z = 2.288, $p = .02$, 95% CI [21.6, 119.79]. Following this, the three predictors (SNA, recalled symptoms and response expectancies) were then inserted into the model simultaneously, to determine their combined effects in predicting DSC. This resulted in the random effect for recalled symptoms becoming nonsignificant, Wald Z = 1.848, $p = .07$, 95% CI [10.98, 91.58], and it was dropped from the model. As *Table 4* model 3 indicates, the fixed effect of each predictor remained significantly related to DSC, and the model fit statistics decreased as compared to the prior two models. Similar to *Model 2*, in *Model 3* the intercept and residual were significant, and most of the variability was within participants; however, the ICC was decreased, ICC= .395. This suggests 39.5% of the variance was between-participants.

Demographic effects

Since gender differences in somatic complaints have been consistently found (Charles & Almeida, 2006; Hiller, Rief, and Brähler, 2006; Jorgensen & Richards, 1989; van Wijk et al, 1999; Verbrudge, 1980), further analyses were run to investigate the influence of gender. Specifically, gender was inserted as a main effect in the model, as well as interaction terms with response expectancies, recalled symptoms, and SNA. None of these terms reached significance, $p > .05$.

We further explored the influence of race/ethnicity in predicting DSC. Using the dummy coded race variables discussed in the method section above, race was entered into the analyses as a main effect, each of which contained one degree of freedom. These analyses revealed no significant main effects of any race or ethnicity, $p<.50$. The influence of race/ethnicity was further entered as interactions with the other variables, and two interactive effects were significant. First, a significant interaction was found between Asian race and recalled symptoms, $B = 0.43$, SE = 0.15, t (98.01) = 2.826, $p = .006$, 95% CI [0.13, 0.72]. Furthermore, when this interaction was entered into a combined model, recalled symptoms was no longer a significant predictor, $p> .05$. Additionally, a significant interaction was found between Hispanic ethnicity and SNA, $B = -1.33$, SE = 0.55, t (400.36) = 2.41, $p = .016$, 95% CI [-2.42, -0.24].

An additional four terms were added to the combined model, namely Asian, Asian x recalled symptoms, Hispanic, and Hispanic x SNA. Reference to *Table* 4 Model 4 indicates that when these four terms were inserted into the model, the model fit criteria were the lower than the prior three models. Additionally, perusal of *Model 4* again shows the residual and intercept were significant and most of the variance was within-participant. After adding the demographic terms

and interactions, the ICC decreased to.359, suggesting 35.9% of the variance was between-participants.

Discussion

This is the first study to simultaneously investigate response expectancy theory (Kirsch, 1985, 1997) and the symptom perception hypothesis (Watson and Pennebaker, 1989) within the context of normal, daily life. Results provide support for both theories. As participants reported higher levels of SNA they tended to report higher levels of DSC. Thus, these results provide support the symptom perception hypotheses.

As per response expectancy theory, participants who reported higher levels of response expectancies reported greater DSC. This relationship appears to have been specific to the items on the CHIPS, as the foil item did not predict DSC, and is consistent with prior findings (Montgomery and Bovbjerg, 2004). Furthermore, this study was a rather conservative test of the effect of response expectancies on DSC. More specifically, after controlling for both SNA and recalled symptoms, response expectancies still exhibited a significant effect. This speaks to the power of response expectancies in predicting somatic complaints. Particularly intriguing was that, when combined, both response expectancies and SNA remained significant predictors of DSC. Additionally, the results of this study did not support the hypothesis that response expectancies moderated the relationship between SNA and DSC. Taken together, these results suggest the effects of SNA and response expectancies on DSC are additive, thus supporting the presumption that the information is incorporated into somatic stimulation from different areas of the brain.

This study also undertook exploratory analyses related to recalled symptoms. The results suggest a positive relationship between recalled symptoms and response expectancies, as well as between recalled symptoms and DSC. Additionally, when a model was created which used SNA, recalled symptoms and response expectancies to predict DSC, all predictors remained significant. As previously stated, this supports the additive effects of SNA, recalled symptoms, and response expectancies in predicting DSC. This study was the first study to integrate response expectancy, recalled symptoms, and SNA in predicting DSC.

A few noteworthy demographic results were found. First, the relationship between some psychological factors and daily somatic complaints seems to differ based on race and ethnicity. The results of the present study suggest that recalled symptoms may only predict daily somatic complaints for individuals of Asian descent. Additionally, the relationship between SNA and DSC was decreased for participants of Hispanic descent. This may reflect cultural differences in the conceptualization of interoceptive experiences. However, caution should be taken as these results need replication. In the current study there was a low number of Hispanic (n = 6) and Asian (n = 13) participants, and the results may be due to some common factor within the participants of these groups that was not measured.

Another notable demographic effect was that analyses did not reveal a relationship between age and DSC; however, as previously discussed, the sample was composed of healthy, young college students. This was intended to avoid this confounding effects due to age. As such, this result was not surprising. Future research should extend the following results to a broader age group, as well as population by using a more representative sample. This would allow for greater generalizability of the findings to a broader population.

This study has a number of additional strengths. This study used daily reports of negative affect and symptoms, which have several advantages over retrospective reports including decreasing recall bias, increased in external validity, and capturing within- and between-person variability (Gunthert & Wenze, 2012). This allowed for the analysis of within-and between-person effects. Furthermore, the use of diaries allow for the examination of mood and daily somatic complaints which had greater generalizability because it was recorded in the participants natural environment (Reis, 1994). Since the data contained within and between-person data, it was analyzed using MLM. MLM has been found to provide more accurate parameter estimates, as well as decreased likelihood of Type I errors. As such, this study was designed to extend previous research into daily life, and provide accurate parameter estimates for this data.

Future Directions and Limitations

Results suggest that recalled symptoms were related to both response expectancies and DSC; however, the results do not support recalled symptoms as a third variable driving the relationship between response expectancies and DSC. Two possible explanations can be generated from the current findings. First, recalled symptoms may represent recollection of actually experienced somatic complaints. Conversely, recalled symptoms may reflect recall bias. More specifically, biased recall may lead to over- or under-reporting of actually experienced symptoms and this recall bias may be the factor driving the relationship with DSC (Gunthert & Wenze, 2012). Due to the research questions of this study though, the authors did not examine if recall bias or experienced symptoms was driving this relationship. Future research could parse out such influences by analyzing recalled symptoms and experienced symptoms, simultaneously. For instance, daily diaries could be used to measure experienced symptoms for a given time period, followed by measuring participants recalled symptoms over the same period of time.

Another diary reporting phase could then be conducted to collect DSC to be used as outcome variables. Thus, researchers would have a measure of experienced and recalled symptoms over the same period of time, allowing them to parse out the contributions of experience and recall bias.

An additional limitation is that participant burden may have precluded some individuals from completing the study, possibly impacting results; however, data analyses did not support this, as excluded and included participants did not differ in recalled symptoms. Additionally, response expectancies were measured as a generalized variable due to prior research suggesting expectancies are stable constructs (Catanzaro & Mearns, 1990), since we were examining subjective somatic information, and to not place further burden on participants. It may be that response expectancies exhibits greater power under situational cues (e.g., encounter with a stimulus believed to provoke a symptom), and as such future research could investigate the within-person variability of response expectancies and possibly its influences as a level one variable. As discussed, similar research has demonstrated that SNA is a stronger predictor of DSC than trait negative affect. A similar relationship may be found between generalized response expectancies and situational response expectancies, provided within-person variability exists. Furthermore, multiple within-day assessments would allow for more discriminate analysis of temporal relationships. As such, assessing factors multiple times a day would not only decrease recall bias, but also allow for analysis of within-day lag effects.

A further limitation was that analyses were conducted at a molar level, as this study focused on the predictions of general somatic complaints. To reflect this aim, a measure (CHIPS) was chosen that sampled several somatic complaints, but did not provide subscales related to specific bodily symptoms (e.g., gastrointestinal). Thus, we did not conduct analyses related to

discrete symptom complaints or bodily systems. Prior research suggests that psychological constructs may differ in their influences related to specific symptoms and/or bodily systems (Charles and Almeida, 2006). Furthermore, the literature suggests that components of negative affect and differing cognitive mechanisms influence somatic complaints in different ways (e.g., recall bias vs. concurrent reports; Howren & Suls, 2011). Future research should be conducted to parse out the differential influences of components of NA with discrete symptom complaints and response expectancies.

Somatic complaints are a common (U.S. Dept. of Health, 2009a, 2009b) and can be a costly issue (Hiller et al, 2006; Kroenke, 2003; Kroenke & Mangelsdorff, 1989; Kroenke et al., 1994; Smith et al, 1986; Stanley et al., 2002). This study integrated two theories relating psychological constructs, response expectancies and negative affect, to somatic complaints. Additionally, this is the first study to examine the influences of recalled symptoms in daily life. The results suggest additive effects of negative affect, response expectancies, and recalled symptoms, as well as suggesting demographic variables influence relationships with daily somatic complaints. Thus, the results of this study suggest that primary care providers should consider a number of variables when patients present with somatic complaints. Such information may lead to improve medical decision making related to testing and treatment, as well as reduced costs and suffering.

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Table 1

Multilevel model specifications used to generate models predicting DSC in SPSS

Specification	Value
Repeated measures covariance	Autoregressive
Random effects covariance	Variance Components
Maximum iterations	100
Maximum step-halvings	10
Maximum scoring steps	1
Singularity tolerance	0.000000000001
Hessian Convergence	0
Log-likelihood Convergence	0
Parameter Convergence	0.000001
Method	REML

Table 2

Bivariate correlations and descriptive statistics for study variables

		1	2	3	4
1	RS	-			
2	RE	.61**	-		
3	SNA	.30*	.37**	-	
4	DSC	.60**	.65**	.56**	-
Mean		27.81	29.38	16.84	15.80
SD		15.94	17.86	5.81	14.11

*p<.01, ***p<.001

Note: Level 1 variables (SNA and DSC) statistics are aggregated within participant; SNA – state negative affect; RS = recalled symptoms; RE – response expectancies

Table 3
Within-person correlations of state negative affect and daily somatic complaints

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Day 1	-	.82*	.47*	.46*	.59*	.77*	.99*
Day 2	.86*	-	.39*	.42*	.57*	.62*	.47*
Day 3	.54*	.67*	-	.53*	.51*	.54*	.46*
Day 4	.50*	.65*	.84*	-	.65*	.22	.20
Day 5	.51*	.59*	.83*	56*	-	.70*	.66*
Day 6	.55*	.73*	.80*	.82*	.82*	-	.73*
Day 7	.57*	.63*	.77*	.76*	.68*	.70*	-

Results above the diagonals represents the correlations for state negative affect (SNA). Results below the diagonals represent the correlations for daily somatic complaints (DSC).
*$p < .05$

Table 4

Fixed Effects Estimates (Top), Variance-Covariance Estimates (Middle), and Model Fit Criteria (Bottom) of the Predictors of Daily Somatic Complaints

Parameter	Model 1	Model 2	Model 3	Model 4
		Fixed Effects		
Intercept	15.70** (1.42) [12.88, 18.53]	9.67** (1.32) [7.04, 12.29]	11.47** (1.32) [8.84, 14.10]	10.86** (1.30) [8.28, 13.44]
Level 1				
SNA		.46** (.09) [0.28, 0.66]	.47** (.10) [0.28, 0.66]	.51** (.10) [0.32, 0.70]
Level 2				
RE		.50** (.06) [0.38, 0.63]	.34** (.07) [.20, 0.48]	.39** (.08) [0.24, 0.54]
RS			.30**(.07) [0.14, 0.46]	.16 (.09) [-0.02, 0.34]
Asian				.82 (2.81) [-4.77, 6.40]
Hispanic				-4.24 (3.95) [-12.03-3.55]
Interaction Terms				
Asian x RS				.45* (.15) [0.15, 0.74]
SNA x Hispanic				-1.36* (.55) [-2.45, -.027]
		Random Effects		
Intercept	149.0**(30.06) [100.34-221.28]	75.48**(18.91) [46.19-123.34]	59.86*(17.41) [33.84-105.87]	51.37* (16.90) [26.96 – 97.88]
AR rho	.55 **(.07) [.40, .67]	.54** (.07) [.39, .66]	.54**(.07) [.39, .67]	.55** (.07) [.40. .67]
AR Diagonal	97.84** (14.28) [73.50, 130.25]	90.34**(12.67) [66.63, 118.92]	91.56** (13.11) [69.15, 121.23]	91.83** (13.54) [68.79, 122.60]

Table 4 (continued)

Parameter	Model 1	Model 2	Model 3	Model 4
		Model Fit Criteria		
AIC	3630.25	3563.05	3553.24	3531.22
BIC	3642.80	3575.60	3565.77	3543.73

Note. Standard errors are in parenthesis and 95% confidence intervals in brackets. **p* <.05, ***p*<.001; SNA – state negative affect; RS = recalled symptoms; RE – response expectancies; AIC – Akaike information criterion; BIC – Bayesian information criterion

Appendix A
CHIPS

HOW MUCH WERE YOU BOTHERED BY:	Not Bothered		↔		Extremely Bothered
1. Sleep Problems (can't fall asleep, wake up in the middle of the night or early in the morning)	0	1	2	3	4
2. Weight Change (gain or loss of 5 lbs. or more)	0	1	2	3	4
3. Back Pain	0	1	2	3	4
4. Constipation	0	1	2	3	4
5. Dizziness	0	1	2	3	4
6. Diarrhea	0	1	2	3	4
7. Faintness	0	1	2	3	4
8. Constant fatigue	0	1	2	3	4
9. Headache	0	1	2	3	4
10. Migraine Headache	0	1	2	3	4
11. Nausea and/or vomiting	0	1	2	3	4
12. Acid stomach or indigestion	0	1	2	3	4
13. Stomach pain (e.g. cramps)	0	1	2	3	4
14. Hot or cold spells	0	1	2	3	4
15. Hands trembling	0	1	2	3	4
16. Heart pounding or racing	0	1	2	3	4
17. Poor appetite	0	1	2	3	4
18. Shortness of breath when not exercising or working hard	0	1	2	3	4
19. Numbness or tingling in parts of your body	0	1	2	3	4
20. Felt weak all over	0	1	2	3	4

21. Pains in heart or chest	0	1	2	3	4
22. Feeling low in energy	0	1	2	3	4
23. Stuffy head or nose	0	1	2	3	4
24. Blurred vision	0	1	2	3	4
25. Muscle tension or soreness	0	1	2	3	4
26. Muscle cramps	0	1	2	3	4
27. Severe aches and pains	0	1	2	3	4
28. Acne	0	1	2	3	4
29. Bruises	0	1	2	3	4
30. Nosebleeds	0	1	2	3	4
31. Pulled (strained) muscles	0	1	2	3	4
32. Pulled (strained) ligaments	0	1	2	3	4
33. Cold or cough	0	1	2	3	4

Appendix B
PANAS

PANAS Questionnaire

This scale consists of a number of words that describe different feelings and emotions. Read each item and then list the number from the scale below next to each word. **Indicate to what extent you feel this way right now, that is, at the present moment *OR* indicate the extent you have felt this way over the past week (circle the instructions you followed when taking this measure)**

1	2	3	4	5
Very Slightly or Not at All	A Little	Moderately	Quite a Bit	Extremely

_____ 1. Interested	_____ 11. Irritable
_____ 2. Distressed	_____ 12. Alert
_____ 3. Excited	_____ 13. Ashamed
_____ 4. Upset	_____ 14. Inspired
_____ 5. Strong	_____ 15. Nervous
_____ 6. Guilty	_____ 16. Determined
_____ 7. Scared	_____ 17. Attentive
_____ 8. Hostile	_____ 18. Jittery
_____ 9. Enthusiastic	_____ 19. Active
_____ 10. Proud	_____ 20. Afraid

VITA

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